EXHIBIT 1

UNITED STATES DISTRICT COURT
EASTERN DISTRICT OF PENNSYLVANIA

IN RE: NATIONAL FOOTBALL LEAGUE PLAYERS' CONCUSSION INJURY LITIGATION	No. 2:12-md-02323-AB MDL No. 2323
Kevin Turner and Shawn Wooden, *on behalf of themselves and others similarly situated*, Plaintiffs, v. National Football League and NFL Properties, LLC, successor-in-interest to NFL Properties, Inc., Defendants.	Civil Action No. 2:14-cv-00029-AB
THIS DOCUMENT RELATES TO: ALL ACTIONS	

DECLARATION OF ROBERT A. STERN, PH.D.

Robert A. Stern, Ph.D., affirms under penalty of perjury the truth of the following facts:

1. I am a Professor of Neurology, Neurosurgery, and Anatomy & Neurobiology at Boston University School of Medicine. My complete *curriculum vitae* is attached at Tab A, and I highlight here some of my experience, research, and qualifications relevant to the opinions expressed below.

2. I am a licensed Clinical Psychologist (Massachusetts License number 7238), with a specialty in Clinical Neuropsychology. I have been licensed as a Clinical Psychologist since 1990 and have been a Registrant of the National Register of Health Service Providers in Psychology since 1992. During that time, I was Director of the Memory and Cognitive Assessment Program at Rhode Island Hospital.

3. Prior to that, I had been Assistant Professor of Psychiatry at the University of North Carolina (UNC) School of Medicine at Chapel Hill, North Carolina, where I had been on the faculty since 1990. During that time, I was Director of the Neurobehavioral Assessment Laboratory as well as the Associate Director of the federally-funded Mental Health Clinical Research Center.

4. I received my Ph.D. in Clinical Psychology from the University of Rhode Island (dissertation titled, "Mood Disorders following Stroke"), completed my pre-doctoral internship training in Clinical Neuropsychology at the Boston Veterans Administration Medical Center, and completed my post-doctoral fellowship research and clinical training in both Neuropsychology and Psychoneuroendocrinology at UNC School of Medicine.

5. I am a Fellow of both the American Neuropsychiatric Association and the National Academy of Neuropsychology. I sit on the editorial boards of several leading medical and scientific journals, and on the grant review committees of several international, national (e.g., National Institutes of Health, NIH), and foundation funding agencies. I am a member of the medical and scientific advisory boards of the MA/NH Chapter of the Alzheimer's Association, the National Grave's Disease Foundation, and Sports Legacy Institute, and am also a member of the Mackey White Traumatic Brain Injury Committee of the National Football League Players Association.

6. Throughout my 25 year career, I have taught medical students and young physicians (neurology residents, psychiatry residents, and geriatrics fellows) through courses and required training seminars in the areas of neurobehavioral mental status examination, brain-behavior relationships, assessment of dementia, the diagnosis and treatment of Alzheimer's disease and related disorders, chronic traumatic encephalopathy (CTE), and similar areas of their formal training.

7. I have been a lecturer in, and a course director of, several continuing medical education (CME) courses for physicians, both locally and nationally.

8. I have been an invited lecturer (and keynote lecturer) for numerous national and international medical and scientific meetings, speaking primarily in the area of Alzheimer's disease, CTE, and issues pertaining to the evaluation and assessment of the cognitive, mood, and behavioral aspects of neurodegenerative disease.

9. I have also been the mentor for numerous undergraduate students, graduate students (Ph.D. students, master's degree students, medical students, M.D./Ph.D. students), and post-doctoral fellows, and have been the primary mentor of many masters theses and Ph.D. dissertations.

10. One of my areas of specialization and expertise includes the assessment and evaluation of neurocognitive functioning. I have published extensively in this area and have also been the primary author of several widely used, standardized neuropsychological tests, including the 33 tests of memory, language, attention, executive functioning, and spatial skills that make up the *Neuropsychological Assessment Battery* (NAB).

11. I have directed predoctoral and postdoctoral training programs in Clinical Neuropsychology, and have served as the mentor for numerous trainees learning to become neuropsychologists.

12. I have given invited lectures at the New York Academy of Sciences and for the Coalition Against Major Diseases in Washington, DC, providing guidance and education to members of the Federal Drug Administration, senior thought leaders in the pharmaceutical industry, and fellow scientists about neurocognitive assessment issues for Alzheimer's disease clinical trials.

13. As a clinical neuropsychologist with a specialty in the evaluation and diagnosis of neurodegenerative diseases, I conduct clinical examinations of patients referred to me by neurologists, geriatricians, psychiatrists, primary care physicians, and others, for diagnostic impressions and treatment recommendations.

14. My clinical neuroscience research focuses on the risk factors for, and the diagnosis and treatment of, neurodegenerative diseases and other causes of cognitive, mood, and behavior change in aging. Currently, I am the Clinical Core Director of the Boston University (BU) Alzheimer's Disease Center (ADC), one of 27 research centers across the country funded by the National Institute on Aging (NIA) of the National Institutes of Health (NIH). In this capacity, I oversee all clinical research (i.e., research conducted on living humans) pertaining to Alzheimer's disease, including studies aimed at the early diagnosis of Alzheimer's disease, genetics, and clinical trials of new medicines to prevent or treat Alzheimer's disease.

15. As part of my role as Clinical Core Director of the BU ADC, I oversee a weekly multidisciplinary diagnostic consensus conference involving neurologists, neuropsychologists, psychiatrists, geriatricians, and others, at which we review the histories, medical tests (including neuroimaging), clinical evaluations, and neuropsychological test performance of research participants and determine the specific

diagnosis (e.g., Alzheimer's disease dementia, Frontotemporal Dementia, Vascular Dementia, Mild Cognitive Impairment, Chronic Traumatic Encephalopathy) of each individual.

16. My other area of currently NIH-funded research includes the cognitive effects of chemotherapy in older breast cancer patients; my co-principal investigators on this grant are from the Georgetown Lombardi Comprehensive Cancer Center and the Memorial Sloan Kettering Cancer Center.

17. Since 2008, my primary area of research has been the long-term consequences of repetitive brain trauma in athletes (see listing of publications below). I was a co-founder of the BU Center for the Study of Traumatic Encephalopathy (CSTE, now "CTE Center") and I serve as the leader of clinical research for the CTE Center. I have received R01 grant funding from NIH (the first grant ever funded by NIH for the study of CTE) to develop biomarkers for the *in vivo* (i.e., during life) detection and diagnosis of CTE.

18. This project, called the Diagnosis and Evaluation of Traumatic Encephalopathy using Clinical Tests (DETECT) study, involves the examination of 100 former professional football players (selected based on positions played, their overall exposure to repetitive brain trauma using data from helmet sensors, and existing clinical symptoms) and 50 same-age non-contact sport elite athletes. All research participants (approximately 100 to date) undergo extensive brain scans, lumbar punctures (to measure proteins in cerebrospinal fluid), electrophysiological studies, blood tests (e.g., for genetic studies and other state-of-the-art biomarkers), and in-depth neurological, neuropsychological, and psychiatric evaluations. For this project, I oversee a talented multidisciplinary group of investigators with specialties in neurology, psychiatry, neuroimaging, radiology, genetics, and biostatistics.

19. In addition, I have recently received Department of Defense funding (with my co-principal investigator, Dr. Martha Shenton from Harvard Medical School) to examine a new Positron Emission Tomography (PET) ligand (T807) that is specific to the abnormal forms of tau protein found in CTE.

20. Relatedly, I am principal investigator of a new study funded by Avid Radiopharmaceuticals to examine that same PET ligand (and another PET ligand for the amyloid protein found in AD) in participants in the DETECT study. I view these two studies of the T807 PET test as the most important investigations in the field of CTE research.

21. I am also the principal investigator of a telephone- and web-based longitudinal study (Longitudinal Evaluation to Gather Evidence of Neurodegenerative Disease; LEGEND) of over 600 adult former and current athletes across all sports and levels of play (including collegiate) to assess risk factors (including brain trauma exposure, genetics, and lifestyle) and clinical course of CTE and other short-term and long-term consequences of repetitive brain trauma.

22. I have conducted over 100 in-depth retrospective clinical interviews with the next-of-kin of the deceased athletes (and others) in Dr. Ann McKee's VA-BU-SLI brain bank. For these cases, I also reviewed all of the available medical records. I currently am a co-investigator of Dr. McKee's NIH-funded U01 project aimed at defining the neuropathology of CTE. For that study, I am a member of the multidisciplinary group of clinicians and scientists who review the clinical history of every new case in the brain bank in order to determine the clinical diagnosis prior to being provided with the neuropathological diagnosis for the case.

23. Based on these experiences, I am confident that I have the same or more experience than any other scientist or clinician in the world examining the clinical history and presentation of athletes (including former NFL players) with post-mortem diagnosed CTE, through detailed interviews and discussions with the decedents' family members, friends, significant others, and physicians.

24. Based on the data gathered through these interviews and medical records, I have published (as first or second author) the largest case series of the clinical presentation of neuropathologically-confirmed CTE (Stern et al., 2013; McKee, Stern, et al., 2013).

25. I am the senior author of an important new journal article (Montenigro et al., 2014) that describes the first clinical diagnostic criteria for CTE and Traumatic Encephalopathy Syndrome (TES), based, in part, on the information gathered from the post-mortem family interviews of over 75 neuropathologically confirmed cases of CTE, and on an extensive review of the world's literature on CTE and "dementia pugilistica."

26. Our group of researchers at BU has been playing a central role nationally and internationally in the area of CTE and the long-term consequences of repetitive brain trauma, including concussions and subconcussive blows. I was the co-director of the first ever national scientific meeting on CTE and have been an invited speaker at numerous national and international conferences, including the first two workshops held

by NIH on this topic. I have published extensively in this area of research including several empirical papers in high impact peer-reviewed scientific journals. I am the invited editor of a special series on CTE and traumatic brain injury (TBI) for the journal, *Alzheimer's Research and Therapy*. I recently testified about this issue before the US Senate Special Committee on Aging.

27. My experience has included extensive clinical- and research-based interviews with former professional football players and their relatives regarding the mid to late life changes in cognition, behavior, mood and daily functioning observed in these persons.

28. My statements and views included in this declaration are mine alone and do not reflect those of Boston University or any of the departments and centers with which I am involved. Specifically, they do not reflect the views of the Boston University Alzheimer's Disease Center, the Boston University CTE Center, or the Boston University Center for the Study of Traumatic Encephalopathy; nor do they reflect any of the faculty, staff, or administration associated with any of these organizations.

29. I have not received any financial payments for preparing this Declaration from any source, including any attorney or plaintiff in this case. Furthermore, I am not retained by, nor receive any payments from plaintiff attorneys in this case for the purpose of this case.

I. CLASS MEMBERS WHO SUFFER FROM MANY OF THE MOST DISTURBING AND DISABLING SYMPTOMS OF CTE WOULD NOT BE COMPENSATED UNDER THE SETTLEMENT

30. I have reviewed the Class Action Settlement Agreement as of June 25, 2014, together with its exhibits (the "Settlement"), filed in the above captioned proceeding. I have paid particular attention to Articles III through IX, and Exhibits 1, 2, and 3, of the Settlement, relating to testing and compensation of the class of retired NFL football players and their families.

31. The primary clinical features of CTE include impaired cognition, mood, and behavior (e.g., Stern et al., 2013). However, the Baseline Neuropsychological Test Battery set forth in Exhibit 2 of the Settlement (the "Test Battery") is focused primarily on the assessment of cognitive impairment, and excludes problems in mood and behavior in the algorithm used to define Neurocognitive Impairment Levels 1, 1.5, or 2.

32. The behavioral and mood disorders associated with head impacts in former professional football players are just as important, just as serious, and just as amenable to detection and diagnosis, as cognitive disorders. Individuals with neuropathologically confirmed CTE have had significant problems with mood and behavior and not just problems with cognition. In the study from my research team (Stern et al., 2013) published in the journal, Neurology, 22 of 33 deceased former athletes with neuropathologically confirmed CTE (and no other abnormal brain findings) were reported to have behavior or mood problems as their initial difficulties, prior to any cognitive impairment. Only 10 of 33 were ever diagnosed with dementia at any time prior to death. These numbers are provided not as an estimate of expected future diagnoses or as an estimate of the prevalence of dementia amongst all individuals with CTE. Rather, they are presented to underscore the findings from our group and from all other descriptions of CTE that dementia and cognitive impairment are not the only life-altering problems experienced by individuals with CTE.

33. Individuals with impairments in mood and behavior, but without significant cognitive impairment can still experience devastating changes in their lives. Based on my review of the medical and scientific literature and on my interviews of living research participants, informal discussions with former players and/or their family members, and formal interviews with family members of deceased former players with neuropathologically confirmed CTE, it is my scientific opinion that many former NFL players have significant changes in mood and behavior (e.g., depression, hopelessness, impulsivity, explosiveness, rage, aggression), resulting, in part, from their repetitive head impacts in the NFL, that have, in turn, led to significant financial, personal, and medical changes, including, but not limited to: the inability to maintain employment, homelessness, social isolation, domestic abuse, divorce, substance abuse, excessive gambling, poor financial decision-making, and death from accidental drug overdose or suicide.

34. The significant changes in mood and behavior relatively early in life can lead to significant distress for the individual with CTE as well as their family, friends, and other loved ones. I have learned about the tremendous pain and suffering the family members experienced while their loved one's life was destroyed by the progressive destruction of the brain. I have interviewed the adult children of former professional and college football and rugby players whose fathers had dramatic changes in personality, the development of

aggressive and out-of-control behavior, and suicidal thoughts. And, I have spoken with the parents of young athletes in their 20's and 30's who impulsively took their own lives.

35. Several well-known former NFL players who were diagnosed neuropathologically with CTE following death did not have dementia and would not have been found impaired under the proposed Baseline Assessment Program of the Settlement (the "BAP"). For example, based on publicly available information, Junior Seau (diagnosed with CTE by a group of independent neuropathologists coordinated by the NIH), and Dave Duerson (diagnosed with CTE by Dr. McKee at BU), both died from suicide reportedly after years of significant changes in mood and behavior, including depression, hopelessness, aggression, and poor impulse control. Based on public reports of their functioning by their family members and friends, it is unlikely that their cognitive skills were impaired to the degree of meeting the criteria for Level 1.5 or Level 2 Neurocognitive Impairment. Rather, their primary symptoms involved mood and behavioral disturbance, neither of which is compensable in the Settlement. Notwithstanding important limitations and criticisms of the test battery and criteria described below, Level 1.5 and Level 2 Neurocognitive Impairment do not include any impairment in mood or behavior. Thus if either of these individuals died on July 8, 2014 or later, their families would not receive any compensation under the Settlement.

36. CTE is a unique neurodegenerative disease. It is not Alzheimer's disease (AD), Parkinson's disease, or ALS. All of these diseases are diagnosed through careful neuropathological examination of brain tissue following death.

37. AD cannot accurately be diagnosed during life, although there have been tremendous strides over the past decade in developing specific, objective biological markers (biomarkers) that improve the predictive accuracy of the diagnosis during life. These biomarkers are now used routinely in research studies and are beginning to be used in clinical settings.

38. CTE also cannot accurately be diagnosed during life, although there are methods being developed at this time by my research team and by others that are meant to improve our ability to do so and to distinguish CTE from AD and other brain diseases and conditions. Based on the scientific and medical literature, my own first-hand knowledge of the current state of the scientific field, and on my own research, I am

confident that within the next five to ten years there will be highly accurate, clinically accepted, and FDA-approved methods to diagnose CTE during life. Based on my involvement in, and understanding of, current ongoing research, it is my scientific opinion that the understanding of neurodegenerative conditions and the capabilities of diagnostic tests will advance rapidly over the next 65 years.

39. Dementia is not an illness or disease. Dementia is a clinical syndrome diagnosed when there are cognitive symptoms that interfere with the ability to function at work or at usual activities, and the patient exhibits a decline from previous levels of functioning that is not explained by delirium or major psychiatric disorder (McKhann et al., 2011; National Institute on Aging and the Alzheimer's Association workgroup).

40. There are several neurodegenerative diseases that can lead to dementia. AD, CTE, and Parkinson's all are neurodegenerative diseases that can lead to dementia. These diseases begin many years or decades prior to any symptoms. When enough brain tissue is destroyed by the disease, symptoms begin to develop. When the symptoms begin, they would not be considered "dementia." When there are cognitive impairments, but not to the degree of interfering with daily functioning, the clinical syndrome of Mild Cognitive Impairment (MCI) may be diagnosed; MCI is not a disease, it is merely a clinical syndrome. It is only when these diseases progress further and the symptoms become bad enough to interfere with the ability to function independently that the individual would be diagnosed with dementia. That is, AD, CTE, and Parkinson's disease each are independent brain diseases that eventually can lead to dementia, later in the course of the disease.

41. The only symptoms related to CTE that are compensable (other than those that overlap with Alzheimer's disease, ALS or Parkinson's) are cognitive difficulties, and only cognitive difficulties that are severe enough that the Class Member would have significant impairments in critical aspects of daily living and independence. Several key symptoms of CTE that are identified in the scientific and medical literature and in my clinical and research experience are not compensable.

42. Class members who clearly have dementia but whose doctors have determined, by appropriate and currently approved medical tests, that they likely have CTE and not Alzheimer's disease as the cause of the dementia would receive substantially less compensation than Class members whose doctors do not order the

tests to assist in the diagnosis. At this time, there are two U.S. Food and Drug Administration (FDA)-approved PET scan tests for patients being evaluated for Alzheimer's disease and dementia: Amyvid (Florbetapir F 18 injection) and Vizamyl (flutemetamol F 18 injection). The following is from an FDA Press Release dated October 25, 2013: "'Many Americans are evaluated every year to determine the cause of diminishing neurologic functions, such as memory and judgment, that raise the possibility of Alzheimer's disease,' said Shaw Chen, M.D., deputy director of the Office of Drug Evaluation IV in the FDA's Center for Drug Evaluation and Research. 'Imaging drugs like Vizamyl provide physicians with important tools to help evaluate patients for AD and dementia…A negative Vizamyl scan means that there is little or no beta amyloid accumulation in the brain and the cause of the dementia is probably not due to AD.'"

As an exemplar, I will compare two hypothetical cases, both age 62 with the same number of qualifying seasons in the NFL. They both have had a progressive history of cognitive, behavioral, and mood symptoms and are now having difficulties carrying out daily activities. They receive the exact same test scores on the Neuropsychological Test Battery and meet the criteria for Neurocognitive Impairment 1.5. They are examined by two different neurologists. Both neurologists conduct neurological evaluations, order the blood tests, and order the same MRI scans. The findings of all these tests come back similarly negative. Both cases are diagnosed by their neurologists as having "dementia." However, Case A's neurologist diagnoses him with Alzheimer's disease. Case B's neurologist decides to order a Florbetapir (Amyvid) PET scan. That specific FDA-approved test is labeled by the FDA to be used to help rule out Alzheimer's disease in cases when the differential diagnosis may be questionable. That is, if the test is found to be negative (indicating little or no abnormal beta amyloid protein build up in the brain), the patient unlikely has Alzheimer's disease as the cause of their dementia. For Case B, because the neurologist knew that CTE was a possible cause for dementia in an individual with a history of repetitive brain trauma, the neurologist felt that the Florbetapir PET scan would be helpful in clarifying the diagnosis. The result of the scan came back negative, resulting in the neurologist determining that the patient does not have Alzheimer's disease. Case A, with a diagnosis of Alzheimer's disease as the cause of dementia, would be eligible for compensation of $950,000 according to the Settlement's Monetary Award Grid. Case B, with a diagnosis of Probable CTE as the cause of dementia (the neurologist

could not give a diagnosis of Alzheimer's based on the negative Florbetapir scan), would not be covered for anything other than Neurocognitive Impairment Level 1.5 and would be eligible for compensation of $290,000. That is, two individuals with identical histories and clinical presentations would receive strikingly disparate compensation solely because of the decision of one of the neurologists to use a very appropriate, FDA-approved test to make a more accurate diagnosis (i.e., not Alzheimer's disease). The former NFL player who received that accurate diagnosis would receive $660,000 less than the former NFL player with the imprecise/incomplete diagnosis.

II. THE BASELINE NEUROPSYCHOLOGICAL TEST BATTERY IS INAPPROPRIATE FOR THE EVALUATION OF THE CLASS MEMBERS FOR WHOM IT IS MEANT TO BE USED

43. The Test Battery, set forth in Exhibit 2 of the Settlement, is not appropriate for evaluating whether retired professional football players have neurodegenerative diseases such as CTE or Alzheimer's disease. Rather, it is appropriate only for the evaluation of a younger traumatic brain injury patient. The specific tests selected, and the length of the battery would not be consistent with that given by the large majority of neuropsychologists who specialize in neurodegenerative disease and who evaluate patients for Mild Cognitive Impairment and Alzheimer's disease dementia.

44. Based on information provided by the test publishers and by my extensive clinical experience with dementia patients, it is estimated that the Test Battery in the Settlement would take approximately five hours without any break. For patients with the level of severity required for compensation (i.e., Level 1.5 or 2 Neurocognitive Impairment), this length of testing would be excessive, would result in refusals to complete the evaluation, and would result in inaccurate results.

45. The Test Battery includes two measures of "Mental Health" even though the results of those tests are not included anywhere in the criteria for impairment. In addition, based on the scientific and medical literature and on my clinical and research experience, the two tests are not appropriate for the detection and diagnosis of the specific types of behavioral and mood disorders linked to a history of head impacts in former professional football players. One of these two tests, the Mini International Neuropsychiatric Interview (M.I.N.I.), is not sufficient to evaluate specific areas of impairments, such as impulsivity, rage, and aggression.

Further, its inclusion in the battery is unnecessary because the results are not used in any way to determine compensable diagnosis. The second of these tests, the MMPI-2RF, is inappropriate for patients with dementia. Even if the results were to be used for any reason, they would likely be inaccurate or incomplete in that the test requires the patient to complete 338 yes-no questions about psychological state and personality; such a task would not be possible by the majority of patients with the severity of dementia included in the compensable diagnoses. As described above, it is my opinion that there must be an appropriate evaluation of mood and behavioral impairment as part of the BAP evaluation, and in the proposed Settlement none exists and there is no inclusion of any mood or behavioral impairment in the definitions of compensable diagnoses.

46. The Test Battery includes extensive testing for performance validity in order to assure that the Class Member's test data represent a valid reflection of the former player's optimal level of functioning, even though patients with moderate dementia have been found to perform poorly (i.e., false positives) on effort testing. Although it is appropriate to consider suboptimal effort in any neuropsychological evaluation for possible compensation, it should be noted that the only compensable findings of the evaluation are Level 1.5 and 2 Neurocognitive Impairment. These represent mild to moderate stages of dementia and require significant impairment on numerous tests in the battery. There have been several studies that indicate that recommended cut-off scores on at least one of the effort tests included in the battery (Test of Memory Malingering) are not appropriate for use in patients with dementia due to an excessive number of false positives (e.g., Bortnick et al., 2013; Teichner & Wagner, 2004). That is, because patients with dementia are so impaired cognitively, they may perform poorly on the effort test due to their actual cognitive impairment rather than poor effort or malingering. It is my scientific opinion, based on the medical and scientific literature and on my own clinical and research experience, that reliance on the effort measures included in the Neuropsychological Test Battery would unfairly deprive at least some otherwise eligible persons with measurable cognitive deficits of compensation.

III. THE HIGH THRESHOLD FOR COMPENSATION BASED ON LEVEL OF COGNITIVE IMPAIRMENT DEFINED BY THE SPECIFIC TEST FINDINGS AND ALGORITHM DETAILED IN EXHIBIT 2 OF THE SETTLEMENT WOULD DEPRIVE PERSONS WITH DOCUMENTED COGNITIVE DEFICITS OF COMPENSATION.

47. To be eligible for compensation under Neurocognitive Impairment Level 1.5 or 2.0, the Class Member would have to be so severely impaired in several areas of cognitive functioning that they would require assistance in many activities of daily living (in Level 1.5) or be almost fully dependent on another person for most activities of daily living, such as bathing and toileting (for Level 2.0). Specifically, the definitions of Level 1.5 Neurocognitive Impairment and Level 2 Neurocognitive impairment require that the Class Member exhibits functional impairment consistent with the criteria set forth in the National Alzheimer's Coordinating Center's (NACC) Clinical Dementia Rating (CDR) scale. For Level 1.5 Neurocognitive Impairment, the Class Member must meet criteria for CDR Category 1.0 in the areas of Community Affairs, Home & Hobbies, and Personal Care. For Level 2 Neurocognitive Impairment, the Class Member must meet criteria for CDR Category 2.0 in the areas of Community Affairs, Home & Hobbies, and Personal Care. According to the CDR, Category 1.0 would require the individual to be unable to function independently at a job, shopping, and volunteer and social groups; to have mild but definite impairment in functioning independently at home, with more difficult chores abandoned, and more complicated hobbies and interests abandoned; and would need prompting for personal care functions, such as dressing, toileting, and bathing. CDR Category 2.0 would require the individual to have no pretense of independent functioning outside home; would only have simple chores preserved; would have very restricted interests; and would require assistance in dressing, hygiene, and keeping of personal effects (Morris, 1993; NACC, https://www.alz.washington.edu/NONMEMBER/UDS/DOCS/VER2/ivpguide.pdf).

48. The algorithm used in the Settlement to translate test performance into compensable Neurocognitive Impairment categories is not one that is used in any known or published set of criteria for the determination of dementia, and utilizes a threshold of impairment that would exclude many Class Members with dementia. To clarify the specific content of Exhibit 2 of the Settlement and understand the algorithm used, it is important to understand the statistical terminology used in the criteria. Neuropsychological tests are developed to result in test scores that are roughly distributed as a normal ("bell-shaped") curve. The tests are typically standardized on a large group of healthy individuals who do not have any known neurological disorder or other possible cause of cognitive impairment. That "normative group" is made up of individuals across

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different age and educational levels, as well as gender and sometimes ethnic, racial, and geographical groups. The results of the normative group's performance on the test are used to create standardized scores, such that when the test is administered to a patient (or in this case a former NFL player), that person's raw score (e.g., the number correct or the time to completion) is compared to the scores from the appropriate reference group from the normative sample. The raw score is then transformed to a standardized score that is then used to interpret the level of performance.

49. A T score is one of the types of standardized scores used to determine the level of performance on the test by the patient. A T score has a mean of 50 (i.e., the average score of the reference normative group is 50) and a standard deviation of 10. A standard deviation is a measure of the distribution of scores in the normative group, such that approximately 68% of the normative group scored within one standard deviation of the mean. That translates into 68% of the healthy normative group having T scores between 40 and 60. Another way to interpret this is that a T score of 40 would be equivalent to approximately the 16th percentile, i.e., only 16 percent of the "normal" healthy population would be expected to score below that level. A T score of 30 (i.e., two standard deviations below the mean) would indicate that only 2.3 percent of the healthy population would be expected to score below that level.

50. As described in Exhibit 2 of the Settlement, the "basic principle for defining impairment on testing is that there must be a pattern of performance that is approximately … 1.7-1.8 standard deviations (for Level 1.5 Impairment) or 2 standard deviations (for Level 2 Impairment) below the person's expected level of premorbid functioning." (Settlement, Exhibit 2, p. 5). Using the tables provided in Exhibit 2 of the Settlement, a Class Member with Average Estimated Intellectual Functioning, for example, would be required to perform worse than 97 percent of same age peers in the published normative reference group on two or more (of six) Learning and Memory tests AND two or more (of four) Executive Function tests, in order to qualify for benefits under the Settlement. As seen in several studies comparing cognitively healthy elderly controls with patients diagnosed with moderate dementia, and often even with severe dementia, it is not common for dementia patients to score consistently more than two standard deviations below healthy controls (e.g., Caccappolo-Van Vliet et al., 2003; de Jager et al., 2003). However, the criteria used in the Settlement would require that the

Class Member's test performance be even more impaired than what is often seen in well-diagnosed cases of moderate stage dementia.

51. The algorithm used to translate test performance into compensable Neurocognitive Impairment categories is arbitrary, nonstandard, and not supported by any scientific literature. There are three different tables in Exhibit 2 of the Settlement used to determine the specific levels of test performance required to meet the categories of Neurocognitive Impairment based on three different levels of "Estimated Intellectual Functioning." That is, the specific number of impaired tests per cognitive domain (e.g., 3 or more versus 2 or more) and the specific level of impairment (e.g., T Score below 35 versus below 37) is different based on whether a Class Member is determined to have Below Average, Average, or Above Average Estimated Intellectual Functioning. Although it is common practice in neuropsychological assessment to compare an individual's performance to expected premorbid levels for that individual, it is uncommon to create distinct criteria tables for levels of impairment based on a single estimate of premorbid functioning to be used across large groups of individuals. And, most importantly, for an algorithm to be used for any decision-making purpose (e.g., determination of large sums of compensation), it must be shown to be valid and reliable in the specific population for which it is being used, a process that requires extensive research. There is no mention in the description of this algorithm that it has undergone any research to determine its appropriateness for this use.

52. As defined in Exhibit 2 of the Settlement, Estimated Premorbid Intellectual Ability is determined by the Test of Premorbid Functioning (TOPF), which "provides three models for predicting premorbid functioning: (a) demographics only, (b) TOPF only, and (c) combined demographics and TOPF prediction equations" (Settlement Exhibit 2, p. 4).

53. Based on the TOPF, Class Members would be categorized into one of the following three categories of Estimated Intellectual Functioning: (1) Below Average (estimated IQ below 90); (2) Average (estimated IQ between 90 and 109); and (3) Above Average (estimated IQ above 110). A Class Member who, based solely on the TOPF predictions of premorbid functioning, is in the Below Average category would have to perform more poorly on more tests than a Class Member who is in the Average or Above Average categories. As an additional exemplar, I will compare two hypothetical cases who receive the exact same test scores on the

Neuropsychological Test Battery with the exception of TOPF scores. Based on the TOPF, the first case would be classified as having Below Average Estimated Intellectual Functioning, whereas the second case would be classified as having Above Average Estimated Intellectual Functioning. The age of both cases is the same, as is the number of qualifying seasons in the NFL. In both cases, the two worse areas of performance are in Learning and Memory and Executive Function. Both cases had two Learning and Memory tests with T scores of 34 and one Learning and Memory test with a T score of 36; all other Learning and Memory tests had better scores (i.e., T scores above 37). Both cases also had two Executive Function tests with T scores of 35 and one Executive Function test with a T score of 36; all other Executive Function tests had better scores (i.e., T scores above 40). Therefore, with the exact same performance on the exact same tests (other than the TOPF word pronunciation test), the first case would not qualify for any compensable diagnosis, whereas the second case would qualify for financial compensation with a diagnosis of Level 1.5 Neurocognitive Impairment.

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Pursuant to 28 U.S.C. § 1746, I state under penalty of perjury that the foregoing is true and correct:

Robert A. Stern, Ph.D.

Date: October 6, 2014